Modern Concepts of Cardiovascular Disease

Published monthly by the AMERICAN HEART ASSOCIATION
44 EAST 23RD STREET, NEW YORK 10, NEW YORK

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Vol. XXVII **JULY, 1958** **No. 7**

BUNDLE-BRANCH BLOCK*

Introduction

During the normal excitation of the heart, electrical activation, after originating in the sinus node, passes over the muscle of the atria in a simple, wave-like fashion and thence through the A.V. node (where the impulse passes rather slowly) into the main stem of the His bundle. After the excitation wave reaches the bundle of His, it passes very rapidly through this structure and then through its right and left branches and the Purkinje network, which ramifies over much of the endocardial surface of the ventricles. These special conducting pathways within the ventricles lead to nearly simultaneous excitation, resulting in properly coordinated and efficient contraction of the right and left ventricles. Since normal excitation occurs from within outward in the ventricles, the deep muscle spirals must contract a little earlier than those situated more superficially. This sequential contraction, with early shortening of the papillary muscles, must be important in expulsion of blood through the normal outflow tracts, with no eversion of, or leakage through, the mitral and tricuspid valves.

The passage of the excitation wave over the atrial muscle causes the P wave in the electrocardiogram; the QRS complex is due to activation of the interventricular septum and the free walls of the ventricles. Evidence of excitation of the special conducting pathways is not seen in ordinary electrocardiograms, because the amount of muscle involved is small and the very rapid passage of the activation wave over these structures would require special leads and equipment for its registration. The T wave of the electrocardiogram represents the electrical recovery or repolarization in the ventricular muscle, but this does not concern us primarily in the present discussion.

If a lesion of any kind (*i.e.*, acute inflammation, neoplasm, fibrosis due to vascular disease, etc.) is suitably located in the interventricular septum, impulses passing through either the main right or left branch of the His bundle will be interrupted and complete right or left bundle-branch block respectively will result. If such a lesion is small or is situated in such a manner that one of the bundle branches is only partially or minimally involved, impulses passing through the branch in question may not be completely blocked, but only delayed. Under these circumstances, incomplete right or left branch block will be present. It should be emphasized that a *single lesion* situated in the right place in the septum may cause bundle-branch block. In numerous patients, whose electrocardiograms show this conduction defect, there are many lesions in the myocardium, but this is not always true and bundle-branch block does not necessarily mean significant myocardial disease.

Anatomical and Physiological Considerations

The main stem of the His bundle passes anteriorly over the upper edge of the interventricular septum, dividing, close to the aorta, into a right and left branch. The former does not further divide into branches that terminate in the Purkinje network until it reaches the base of the right anterior papillary muscle. The latter separates into two major divisions, anterior and posterior, high on the left side of the septum. These differences in the distribution of the bundle branches explain the physiological, early excitation of the left side of the septum and the well-known fact that left branch block is more likely to be associated with serious heart disease than is right branch block.

With complete right or left bundle-branch block (and to a lesser degree in incomplete branch block) the right and left ventricles, respectively, are excited later than with normal intraventricular conduction, and one might

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therefore expect the left ventricle to contract a little earlier than the right ventricle in right branch and slightly later than the right ventricle in left branch block. If it is agreed that the first heart sound is caused primarily by the closure of the mitral and tricuspid valves and that the second sound is due to the closure of the aortic and pulmonic valves, then it would be anticipated that both the first and second heart sounds would be split in patients with bundle-branch block. This has been understood for many years, but since splitting may occur in the absence of bundle-branch block, its presence was not considered to be a valuable aid in the clinical diagnosis of such block until Leatham[1] clarified the whole matter recently. He re-emphasized that in many subjects with normal intraventricular conduction, splitting of the pulmonic second sound occurs only during or near the end of inspiration, due to a slight delay in the closure of the pulmonic valve, and further that this physiological type of splitting may be recognized by careful auscultation. In patients with complete right branch block, persistent splitting of the pulmonic second sound, becoming somewhat more marked with inspiration, was observed. In those with left branch block, a paradoxical type of splitting in which the pulmonic second sound was single during inspiration, and split during expiration, was found. These interesting studies are of great clinical value, since they make it possible, on purely auscultatory findings, to suspect the presence of right or left branch block. It has been generally assumed for many years that intraventricular block can be diagnosed with certainty only from the electrocardiogram. This statement is still true, in general, but nevertheless the wide awake and interested physician now has an incentive for giving careful attention to the character of the heart sounds, especially the pulmonic second sound. To the physician who suspects bundle-branch block from stethoscopic findings, it is a great source of satisfaction to have this opinion confirmed by an electrocardiogram.

The Electrocardiogram in Bundle-Branch Block

When bundle-branch block is present, excitation of one of the ventricles is delayed. It would, therefore, be anticipated that the entire period of time required for activation of these two chambers, that is the QRS interval, would be increased. Furthermore, the increase in this interval should be greatest in complete branch block and progressively less with decreasing degrees of incomplete block. It should be clear that in the latter situation, when there is only slight delay as excitation passes over one of the bundle branches, the QRS interval may be within normal limits. Under these circumstances, the diagnosis of the conduction defect must depend entirely on changes in the form of the QRS complexes in certain leads and there may be uncertainty regarding the proper interpretation of electrocardiograms of this kind. These matters will be discussed in more detail later in this paper.

Before the electrocardiographic changes occurring with bundle-branch block are discussed further, a few words must be said about other conditions which might be confused with it, because they also are associated with long QRS intervals. First, however, it should be emphasized that the term "bundle-branch block" implies either complete interruption, or delay, of the excitation wave passing through the *main* right or left branch of the His bundle.

Block may occur elsewhere within the ventricles, somtimes involving only localized areas of the left ventricle, as in peri-infarction block. Occasionally, a diffuse process involving the entire myocardium may cause an abnormally long QRS interval. Examples of the latter are seen in hyperkalemia, quinidine intoxication and in dying hearts. Peri-infarction block is usually easily recognized by other changes pointing to myocardial infarction in the electrocardiogram. In this type of block, a definite increase in the QRS interval may be seen in only a few leads. Most of the conditions, like hyperkalemia, that increase the QRS interval by slowing the rate of excitation may be suspected by other changes in the electrocardiogram or by the clinical findings.

Bundle-branch block should not be considered as the cause for an increase in the QRS interval unless it is clear that the ventricles are responding to a pacemaker located *above the bifurcation of the His bundle*. This is usually obvious, but occasionally confusion results when ventricular premature beats arise late in diastole and excitation occurs in the ventricle, both from the ectopic focus and over the normal pathways. Since such a beat may be associated with an abnormal QRS complex and a long QRS interval, it may be wrongly interpreted as being due to transient bundle-branch block. If bilateral complete bundle-branch block is present, complete A.V. heart block will also exist and under these circumstances the true nature of the condition may be obscure. Rosenbaum and Lepeschkin[2] have recently discussed this, and similar conditions, in detail.

Another condition which may simulate bundle-branch block is anomalous A.V. excitation (Wolff-Parkinson-White syndrome). Here the QRS interval is increased into the range common with bundle-branch block, but the P-R interval is correspondingly shortened. Furthermore, the usual iso-electric interval between the end of the P wave and the beginning of the QRS complex is usually absent and an R wave with slurring of the upstroke, and no Q wave,

is seen in leads I, aV_L and chest leads taken over the left ventricle (usually V_4 through V_6). The mechanism responsible for tracings of this kind is not definitely known, but both the short P-R interval and the increase in the QRS interval may be explained by an accessory pathway between the atria and ventricles, which bypasses the usual pathway of excitation through the A.V. node, bundle of His, the bundle branches and the Purkinje network. Although most individuals whose electrocardiograms show this peculiar type of conduction have no other clinical evidence pointing to heart disease, many of them are subject to attacks of rapid heart action, usually supraventricular paroxysmal tachycardia, which may be difficult to control.

Left Bundle-Branch Block. The QRS interval in complete right or left bundle-branch block is usually 0.12 to 0.16 second. This interval tends to be somewhat longer in left as compared with right branch block, probably due, not only to the greater thickness of the wall of the left ventricle, which is usual, but also to left ventricular hypertrophy, often present in patients with left branch block.

One of the most important electrocardiographic changes that occur with left branch block is due to delay in excitation of the left side of the septum. With normal intraventricular conduction, as mentioned earlier, there is early excitation of the left side of this structure, which usually causes the initial electromotive forces to be directed from left to right, and anteriorly. These voltages are unopposed during the first few hundredths of a second (0.01 to 0.03 second) and therefore cause the normal, septal Q waves so frequently seen in leads I, aV_L and in chest leads V_5 and V_6. The initial portions of the early R waves seen in chest leads from the right side of the precordium are also due to these early septal electromotive forces. With the above facts in mind, it is possible to understand why these normal septal Q waves are abolished by both complete and incomplete left branch block. Furthermore, excitation of the septum is grossly changed, so that it is almost entirely from right to left side in complete left branch block and altered in a similar manner but to a lesser degree in incomplete left branch block. Thus, with complete left branch block, the early electromotive forces in excitation of the ventricles (except as may be slightly modified by normal activation of the free wall of the right ventricle) are oriented from right to left, and posteriorly. These electromotive forces cause initial R waves (with no Q) in precordial leads over the left ventricle and in leads I and aV_L, if the heart is in the usual horizontal position. It was pointed out above that the initial part of the R waves seen in precordial tracings taken over the right ventricle in normal intraventricular conduction is due to early excitation of the left side of the septum. When left branch block is present, this component of the R waves is lost and the small narrow R waves, or complete absence of R waves in these leads, are due to weak right ventricular effects or their complete abolition by septal voltages passing in the opposite direction.

The free wall of the left ventricle is excited later than is normal in left branch block. The delay is greatest, of course, in complete left branch block and less in incomplete block. Excitation of this chamber causes voltages oriented from right to left, and posteriorly. These are similar in direction to those passing earlier through the septum in left branch block and cause the final peaks of the broad notched R waves, usually seen in complete block of this kind, in leads I, aV_L and in chest leads V_5 and V_6. Similar findings of lesser degree are seen in incomplete left branch block.

It is not uncommon to see electrocardiograms that resemble those seen with complete left branch block, except that Q waves are present in leads I, aV_L and chest leads from the left side of the precordium. Such tracings are difficult to explain on the basis of simple, uncomplicated left branch block, as should be clear from the discussion above. Many individuals with records of this kind have coronary artery disease with more or less extensive infarction in the free wall of the left ventricle, but these infarcts alone do not explain the Q waves in question, because in left branch block the electromotive forces early in excitation pass from right to left through the septum and cause initial R waves (not Q waves) in the leads mentioned above. If, however, *there is also infarction of the septum,* more or less completely eliminating the muscle responsible for these early right to left voltages, the appearance of Q waves may be understood. This has recently been emphasized by Chapman and Pearce[3], who also have pointed out that for other reasons, left branch block may not wipe out alterations in the QRS deflections due to infarction as completely as has been assumed in the past.

Although there is little experimental or other evidence to support the idea, the writer has wondered if some of the tracings resembling left branch block, except for the presence of Q waves in leads I, aV_L and V_6, may not be caused by block in one of the main subdivisions of the left bundle. Such a lesion might delay excitation of portions of the free wall of the left ventricle sufficiently to increase the QRS interval to a significant extent and not alter septal activation as much as block in the main left branch. The above suggestion is purely speculative at the moment, but might help to explain some tracings resembling left branch block, except for Q waves, in patients who show no clinical evidence suggesting infarction.

The electrocardiographic diagnosis of incomplete left branch block may be difficult, particu-

larly when the QRS interval is not increased to 0.10 second or more. Under these circumstances, the diagnosis must depend almost entirely on the absence of Q waves in leads I, aV_L and chest leads V_5 and V_6. Sodi-Pallares,[4] and associates, have presented experimental and clinical evidence pointing to the importance of these criteria in the recognition of minor degrees of incomplete left branch block; in their opinion, block of this kind is more common than has been generally appreciated. One must remember, however, that Q waves, in the leads mentioned, may be absent as a result of unusual rotation of the heart, particularly about its long axis. The writer is therefore unwilling to make a definite diagnosis of incomplete left branch block unless the QRS interval is at least 0.10 second or there is other evidence (such as a previous tracing showing clear signs of left branch block) which makes the nature of the conduction defect clear.

Finally, the strong resemblance of the ventricular complexes in incomplete left branch block to many tracings showing anomalous A.V. excitation (Wolff-Parkinson-White syndrome) must be mentioned. As mentioned earlier in this paper, the short P-R intervals present in tracings of the latter group usually make it possible to distinguish between these two conditions without difficulty.

Right Bundle-Branch Block. The basic defect in right branch block is, of course, complete interruption of or delay in the passage of the excitation wave through the right branch of the His bundle in complete and incomplete right branch block respectively. This means that excitation of the free wall of the right ventricle occurs later than normal, the delay being maximal in complete block in which the activation occurs after that in the septum and the left ventricle has been largely completed. Under these circumstances, electromotive forces arising in the right ventricle are essentially unopposed and, as would be expected from the anatomical position of this chamber, are directed from left to right, and anteriorly. This means, of course, that the *final deflection* of the QRS complex in precordial leads taken over the right ventricle (usually V_1 and V_2) will be a late R wave. These same voltages also usually cause a final downward deflection (S wave) in chest leads from the left precordium and in leads I and aV_L.

Excitation of the interventricular septum in complete right branch block is probably predominantly from left to right, but since the left side of this structure is excited earlier than the right side in normal intraventricular conduction, the differences in septal activation are not as marked in right branch as they are in left branch block. In any event, the earliest electromotive forces in right branch block (as with normal intraventricular conduction) arise on the left septal surface and spread from left to right, and anteriorly, through this structure. They cause early R waves in chest leads V_1 and V_2 and Q waves in chest leads V_5 and V_6 and in leads I and aV_L.

In the above discussion, little has been said about voltages during excitation of the left ventricle. In both complete and incomplete right branch block, it is likely that septal activation is largely completed before that in the left ventricle is entirely finished. This means that in right branch block there is a temporary reversal in the average direction of excitation between the early, left to right septal voltages and the final electromotive forces passing in the same direction, due to late activation of the right ventricle. Therefore, in chest leads from the right precordium (V_1 and V_2), one would expect M-shaped complexes or initial and final R waves separated by a downward deflection. This downward wave may or may not go below the base line in these leads. These left ventricular voltages, causing the downstroke in question are, of course, also the reason for R waves in chest leads from the left precordium and usually in leads I and aV_L.

With right branch block, the early voltages in ventricular excitation are oriented from left to right. Furthermore, the only muscle whose activation can cause both reversal of this direction and R waves to occur in leads over the left ventricle is situated in this chamber; therefore, neither incomplete or complete right branch block (as may be true in left branch block) prevent broad Q waves or QS waves appearing with myocardial infarction. In other words, the usual QRS changes of myocardial infarction are found in the presence of right branch block much as they are with normal intraventricular conduction.

The presence of both right ventricular hypertrophy and right branch block may be difficult to ascertain with certainty. Under these circumstances, unusually tall late secondary R waves in V_1 are present, as would be expected. The difficulty arises from the fact that the size of these R waves varies greatly when right branch block alone is present.

One of the most troublesome problems encountered in the interpretation of precordial electrocardiograms arises in connection with many tracings in which late secondary R waves (M-shaped QRS complexes) are found in V_1 or V_2, or both. When the QRS interval is 0.10 to 0.12 second, incomplete right branch block is obviously present, but how about the large number of records in which the QRS interval is 0.08—0.10 second, or even somewhat less? Some in the latter group with QRS intervals of approximately 0.09 second may represent minor grades of incomplete right branch block. Many individuals having electrocardiograms of this kind present no other clinical evidences pointing to heart disease, so it is difficult to believe that all

of these electrocardiograms are due to true right branch block. An alternative explanation for late secondary R waves in chest leads V_1 or V_2, when the QRS interval is normal or only slightly increased, may be found in physiological late activation of muscle near the outflow tract of the right ventricle. Supporting this view is the observation that in many normal subjects, late secondary R waves are present in tracings taken along the left sternal border in the second or third intercostal spaces and that in these individuals smaller late R waves may or may not be present in V_1 or V_2. Furthermore, Walker, and associates,[5] have pointed out: (1) that patients with atrial septal defects may be expected to develop hypertrophy of the outflow tract of the right ventricle, and (2) that their electrocardiograms, commonly interpreted as showing right branch block, may not in fact represent true right branch block but show late secondary R waves in V_1 and V_2 as a result of physiological late activation of an hypertrophied outflow tract.

At the present time, there is no general agreement concerning the problems raised in the above paragraph and further work is needed to clarify them. For the moment, it is important to emphasize that whether all of the tracings under discussion are due to right branch block or part of them to right branch block and others to something quite different, many of the patients from whom they are obtained have no organic heart disease and the electrocardiographic peculiarities should not be interpreted as pointing to cardiac abnormality. The same caution should be exercised in cases of definite incomplete right branch block; there are many apparently "normal" individuals with complete right branch block and less commonly with left branch block. When an electrocardiogram, particularly one taken as part of a general examination, shows complete right branch block or either complete or incomplete left branch block, the patient should have a more searching cardiac examination, including a careful history and physical examination of the heart, than would otherwise be done. If these studies fail to reveal any findings pointing to heart disease, the patient should not be told that he (or she) has cardiac abnormality, nor should his life or activities be altered in any way.

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